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Seroepidemiological Survey of Hepatitis B and C Virus Infections in Ghanaian Children

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The seroprevalences of hepatitis B virus (HBV) and hepatitis C virus (HCV) markers were evaluated in a random sample of 803 children attending school in Ashanti-Akim North district in Ghana in order to gain a better understanding of transmission patterns of these viruses, particularly horizontal transmission of HBV. This rural district is typical of 70% of the Ghanaian population. The overall seroprevalence of at least one marker of HBV infection was 61.2%, with rates increasing from 48% to 80% between the ages of 6–18 years ($P < 0.001$). The overall HBsAg seroprevalence was 15.8%, with the proportion of HBsAg positives amongst those with anti-HBc increasing from 39.3% in 6–7-year-olds to 51.8% in 12–13-year-olds. It appears that horizontal transmission during this age period was accompanied by a high rate of HBsAg carriage. Among those infected but not carriers, i.e., those HBsAg negative and anti-HBc positive, >50% lacked detectable levels of anti-HBs, an unusual pattern of convalescent immune response to HBV. The overall seroprevalence of anti-HCV was 5.4% and did not differ significantly by age or gender. Anti-HCV seroprevalence was not associated with the presence of any HBV marker. A better understanding of the unusually high prevalences of HBV and HCV infections demonstrated in this population is likely to influence vaccination and blood transfusion policies and to stimulate further evaluations of these infections and their vehicles of spread in highly endemic regions such as sub-Saharan Africa.

KEY WORDS: HBsAg, anti-HBc, anti-HBs, anti-HCV, Ghana, Africa

INTRODUCTION

Hepatitis B virus (HBV) carriers are estimated to number > 300 million worldwide, representing ~5% of the world's population. Varying levels of endemicity exist throughout the world with highly endemic areas in sub-Saharan Africa, Asia, the Far East, and the Mediterranean basin. In highly endemic regions most transmission is thought to occur during infancy and early childhood. The annual incidence of HBV infection among children has been projected to be 3–5%, with both antigenemia and antibody prevalence increasing up to preadolescent ages and dropping thereafter [Szmuness et al., 1978]. Persistent HBV infections and chronic HBsAg carrier state result more often from infection during infancy and early childhood.

In the sub-Saharan African region, HBsAg carriage rates vary substantially, ranging from 7% to well over 20% [Sobeslavy et al., 1980; Whittle et al., 1983; Tabor et al., 1985]. In the Ashanti-Akim district in Ghana, liver diseases rank fourth as a cause of mortality, accounting for ~8% of deaths [Amonoo-Lartson et al., 1985]. Despite early disease surveys, which indicated a high prevalence of clinical hepatitis [Morrow et al., 1969], no data on the seroprevalence of various hepatitis viruses in Ghana have been published. Although there is a need to include HBV immunization in the list of childhood immunizations for Ghana, inadequate financial resources and a lack of political commitment stemming from the absence of data from Ghana have not made this possible. There is a need to determine the prevalence of HBV infection in Ghana in order to determine the priority to be accorded the addition of HBV immunization to the list of childhood immunizations.

Hepatitis C virus (HCV), which also has a worldwide distribution, is now responsible for the vast majority of posttransfusion non-A, non-B hepatitis infections. However, transmission of HCV also occurs in the absence of obvious parenteral or sexual exposures [Chiaramonte et al., 1991]. Only very limited data are available concerning the prevalence of HCV infection in Africa, and no

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information is available on seroprevalence of HCV in Ghana. Even more than HBV, HCV infections are often persistent and may lead to chronic liver disease. The relatively high rates and severity of anemia from malnutrition, hemoglobinopathies, infections, and infestations in early childhood in West Africa frequently result in repeated blood transfusions in a substantial proportion of these afflicted children. However, the development of assays for detection of anti-HCV has not resulted in the availability of HCV-free donor blood in these areas since the cost of these assays is still beyond the reach of the health care system. As with HBV, it is likely that lack of knowledge concerning the prevalence of HCV infection in these areas and its relation to liver specific morbidity and mortality, has contributed to the present lack of attempts at control of this infection.

We report the results of a study examining the seroprevalence of hepatitis B and C markers in a representative population sample of children in the Ashanti-Akim North district in Ghana. This study provided the opportunity to evaluate simultaneously HCV and HBV seroprevalence in a highly endemic HBV region and identify differences in age-specific seroprevalence rates. Such information will be useful in determining strategies for immunization against HBV and designing educational programs aimed at reduction of risk factors for transmission of both HBV and HCV in this environment. The findings raise some novel questions about the immune response to HBV and horizontally transmitted HBV.

MATERIALS AND METHODS

Study Population

This cross-sectional study was carried out in the Ashanti-Akim North district in Ghana. The district has a population of ~130,000 (projections from 1980 census figures) and is representative of the 70% of Ghanaians estimated to be living in rural districts. Children ≤15 years form ~50% of the population, with >90% of children aged 6–16 years enrolled in school. This district, one of 110 in Ghana, is predominantly rural with only three communities having a population exceeding 10,000. There was no history of HBV immunization in the district when the study was initiated in October 1993.

The study population consisted of children between 6–18 years old who had lived in the district for at least 1 year. Exclusion criteria included being married, lack of consent to participate from parents, and clinical evidence of anemia. For logistic reasons, the study was limited only to the 14 communities with populations of 2,000 or more, which comprise ~73% of the district population, and to children enrolled in school. Within these communities, 810 children were selected from primary schools and junior secondary schools (JSS) out of 18,072 schoolchildren aged 6–18 years in the district. In the first stage of sampling, 40 schools were selected, 20 each from the 44 primary schools, and 26 JSS, using probability proportional to size cluster sampling, stratified by school type. In order to obtain similar numbers of children from each birth cohort, four children were randomly selected from each grade in the second stage of sampling, resulting in 24 pupils being selected from each primary school, and 12 pupils from each JSS. In addition, we selected four pupils from those aged 16 years to 18 years in each JSS. The sampling probability in primary schools was estimated to be 0.036, whereas that in JSS was estimated to be 0.067. Oversampling in JSS provided sufficient sample size for calculation of age-specific seroprevalence rates.

This study was approved by both the Ministry of Health, Ghana, and the Institutional Review Board on Research Involving Human Subjects, School of Public Health, University of North Carolina (Chapel Hill).

Sample Collection

Five field teams were selected from staff of the Ministry of Health within the district. During their initial visit to the selected schools, these teams sampled pupils for inclusion into the study, selected alternates to replace potential refusals, recorded names, gender, age, and addresses of subjects, and provided them with consent forms to be initialed by their parents. During a second school visit, study teams drew 5 ml of venous blood from the antecubital fossa of children of consenting parents. Blood was kept in cold boxes and transported to the study laboratory at the district hospital, where serum was separated within 6 hours of collection and stored at −20°C. Specimens were subsequently shipped on dry ice to the Experimental Biology Department, Abbott Laboratories (Chicago) for identification of HBV and HCV markers.

Serological Tests for HBV and HCV

Serum samples were tested with commercial immunoassay reagents supplied by Abbott Laboratories: AUSRIA II for HBsAg, AUSAB for anti-HBs, and CORAB for anti-HBc. Screening for HCV antibody was carried out using HCV EIA 2.0 with supplemental MATRIX HCV 2.0 and also antibody to hepatitis C virus second envelope (HCV-E2) glycoprotein as described by Lesniewski et al. [1995]. We considered a subject to be positive for HCV if he/she tested positive by HCV EIA 2.0 and either the supplemental MATRIX HCV 2.0 or HEV-E2 glycoprotein assays or both.

Statistical Analyses

All analyses were done using SUDAAN 6.2 software (RTI, Durham, North Carolina), which took into account the sampling design and adjusted variances for seroprevalence accordingly. For each age-specific, gender-specific, and community size-specific seroprevalence, variances and 95% confidence intervals were calculated. Differences in prevalence between genders and between the two defined community sizes were evaluated for significance using Chi-square tests, whereas linear trends in the age-specific seroprevalences were evaluated using the Chi-square test for linear trend.

TABLE I. Seroprevalence of Hepatitis B Virus and Hepatitis C Virus Markers With 95% Confidence Intervals

	n	Any HBV marker (95% CI)	Anti-HBc (95% CI)	Anti-HBs (95% CI)	HBsAg (95% CI)	Anti-HCV (95% CI)
Age (years)						
6–7	98	48.0 (35.8–60.1)	36.7 (26.7–46.7)	21.4 (10.1–32.7)	10.2 (4.9–15.5)	3.1 (−0.3–6.4)
8–9	139	44.6 (34.9–54.3)	38.9 (29.1–48.6)	18.8 (12.8–24.8)	10.1 (5.1–15.0)	6.5 (2.2–10.8)
10–11	154	56.8 (49.2–64.4)	50.5 (44.1–56.9)	22.4 (16.6–28.3)	19.5 (14.5–24.6)	3.3 (0.2–6.4)
12–13	186	77.8 (69.2–86.4)	71.5 (63.1–79.9)	27.1 (20.1–34.0)	20.9 (13.3–28.6)	6.8 (3.1–10.5)
14–15	127	74.9 (66.8–82.9)	71.9 (64.3–79.5)	29.2 (18.0–40.5)	14.6 (6.2–22.9)	8.6 (3.1–14.1)
16–18	99	79.8 (73.9–85.7)	75.8 (68.5–83.0)	35.4 (26.7–44.0)	19.2 (11.9–26.6)	5.1 (0.7–9.4)
Total	803	61.2 (56.6–65.7)	54.8 (51.0–58.7)	24.3 (20.7–27.9)	15.8 (13.0–18.6)	5.4 (3.5–7.3)
P value*		$P < 0.001$	$P < 0.001$	$0.60 < P < 0.70$	$0.05 < P < 0.10$	$0.70 < P < 0.80$
Gender						
Males	392	68.3 (63.9–72.7)	62.1 (57.9–66.2)	24.9 (20.5–29.3)	18.6 (14.6–22.7)	5.0 (2.8–7.3)
Females	411	54.5 (48.4–60.7)	48.1 (42.6–53.5)	23.7 (18.9–28.5)	13.2 (9.4–17.0)	5.8 (2.9–8.6)
P value*		$P < 0.005$	$P < 0.005$	$0.80 < P < 0.90$	$P = 0.07$	$0.60 < P < 0.70$
Community size						
< 10000	305	63.6 (56.6–70.6)	56.8 (50.5–63.1)	25.9 (20.1–31.8)	16.9 (12.7–21.2)	5.0 (2.4–7.5)
> 10000	498	58.9 (52.2–65.7)	53.0 (47.0–59.0)	22.8 (18.5–27.0)	14.8 (11.1–18.4)	5.8 (3.0–8.6)
P value*		$0.15 < P < 0.20$	$0.20 < P < 0.25$	$0.15 < P < 0.20$	$0.15 < P < 0.20$	$0.70 < P < 0.80$

*P value is to test for linear increase with age and for significant difference in seroprevalence rates of antigen and antibodies in the two size strata for gender and community size.

RESULTS

Study Population

Serum samples were evaluated from 803 out of 810 selected pupils. Of the 803 pupils, 392 (49%) were females. More than 30 pupils were included from each birth cohort from 6–16 years of age, with the 17-year and 18-year-old birth cohorts contributing 29 pupils and 14 pupils, respectively. The gender distribution in each birth cohort was about equal. Of the 803 pupils, 305 (38%) were enrolled from communities with populations <10,000 (small communities) and 498 (62%) were from communities with populations of 10,000 or more (large communities). The sampling fraction of pupils from small communities was estimated to be 0.043 compared to 0.046 from large communities.

Hepatitis B Virus

Of these 803 pupils, 61.2% had at least one marker of HBV infection. About 50% of all pupils below 12 years old had such evidence of HBV infection, and this rate rose to >75% in those 12 years old and over. The increase in HBV seroprevalence with age was highly significant ($P < 0.001$, Table I). The overall seroprevalence of anti-HBc was 54.8%, and age-specific seroprevalence of anti-HBc also increased with age ($P < 0.001$). Anti-HBs seroprevalence was 24.3%. Although the seroprevalence of anti-HBs increased with age, this was not significant ($0.60 < P < 0.70$). The seroprevalence of HBsAg was 15.8%, with this trending upward from 10.2% in 6–7-year-olds to 20.9% in 12–13-year-olds, thereafter stabilizing ~20% (Table I).

A consistent trend toward greater prevalence of HBV markers was observed in males than in females (Table I). The prevalence difference between genders was largest when the presence of any marker (68.3% vs. 54.5%, $P < 0.005$), and the prevalence of anti-HBc (62.1% vs. 48.1%, $P < 0.005$) were considered. However, the differences between gender-specific anti-HBs and HBsAg prevalence rates were smaller and not statistically significant. Although the prevalences of HBV markers were consistently higher in pupils from smaller communities, this trend was not statistically significant.

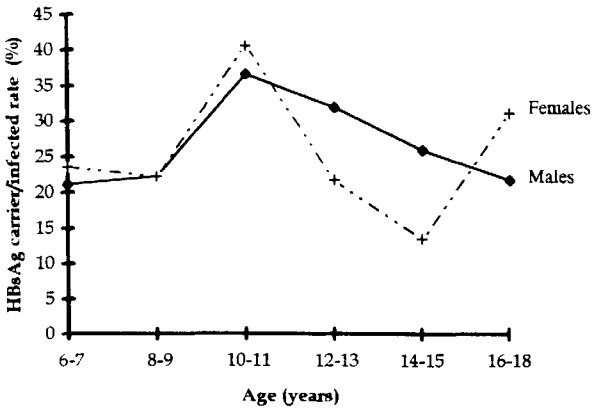

Fig. 1. HBsAg carriage rates among anti-HBc positive school children by gender.

We defined a "carrier/infected rate" as the proportion of HBsAg positive pupils among those who were anti-HBc positive. Overall, this rate was estimated to be 26.9%. However, the "carrier/infected rate" was ~20% in pupils ages 6–9 years, then increased to almost 40% in pupils aged 10–11 years, after which there was a general decline. The "carrier/infected rate" was similar in males and females up to the 10–11-year-old cohort, after which the rate in females dropped faster than in males until the 16–18-year cohort, where the rates in the two genders again approximated each other (Fig. 1). However, the overall "carrier/infected rate" for males was not significantly different from that in females ($0.25 < P < 0.30$).

The prevalence in children with HBsAg as the sole marker of infection was determined, and this rate was

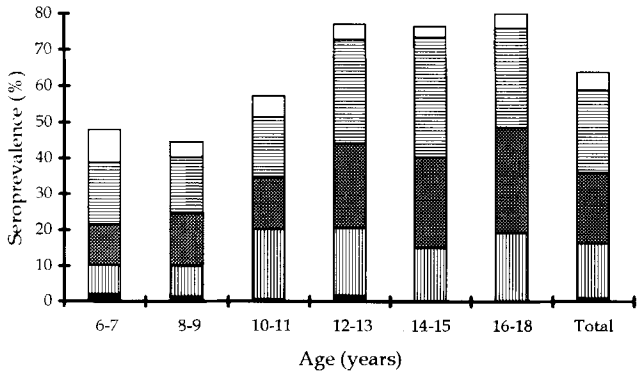

Fig. 2. Seroprevalence of HBV markers in schoolchildren in rural Ghana. Prevalence of five mutually exclusive categories of combinations of HBV markers: ■ HBsAg+ only; ▥ HBsAg+, Anti-HBc+; ▩ HBsAg−, Anti-HBc+, Anti-HBs+; ▤ HBsAg−, Anti-HBc+, Anti-HBs−; □ HBsAg−, Anti-HBc−, Anti-HBs+; are depicted by age group. The categories sum to the age-specific prevalence of any HBV marker.

used as a proxy for the minimum number of persons with new infection across age groups. These rates ranged from 0.6% to 2% in 6- to 13-year-olds, after which the rate was 0% (Fig. 2). We also calculated age-specific rates of pupils who were HBsAg-negative and anti-HBc-positive but had no detectable anti-HBs, i.e., those presumed to have recovered from infection. This proportion was 61–48% across the age groups (Fig. 2).

Hepatitis C Virus

The seroprevalence of anti-HCV was 5.4% overall. Although this seroprevalence showed an increasing trend with age ranging from 3.1% in 6–7-year-olds to a maximum of 8.6% in 14–15-year-olds, these differences were not statistically significant ($0.70 < P < 0.80$, Table I). Unlike HBV marker prevalence, seroprevalence of anti-HCV in males (5.0%) was slightly lower than in females (5.8%). However, this small difference was not significant ($0.60 < P < 0.70$, Table I). Seroprevalence of anti-HCV in small communities (5.0%) was only slightly lower than that in large communities (5.8%) ($0.60 < P < 0.70$, Table I).

Table II shows the distribution of anti-HCV in pupils categorized according to the results of tests for the various HBV markers. Anti-HCV seroprevalence was not associated with the presence of any of the HBV markers.

DISCUSSION

The seroprevalence of any marker of HBV infection was found to be 61.2%, and that of HBsAg to be 15.8% in children aged 6–18 years in Ghana. The seroprevalence of all markers of HBV infection increased with age, with the rate of HBsAg carriage increasing among those infected up to 12–13 years of age, after which there was a gradual decline. There was a significantly higher seroprevalence of anti-HBc among males, but males were no more likely to become carriers of HBV than females. The overall anti-HCV seroprevalence was 5.4%, but in contrast to the HBV markers, there was not a significant gender difference.

This study confirms the high endemicity of HBV in sub-Saharan Africa. The 61.2% prevalence of any HBV marker of infection that we observed was considerably higher than the 40% Tabor et al. (1985) found in Zambian children, but quite comparable to the 58.5% found among children in Cameroon [Chiaramonte et al., 1991]. Comparison of seroprevalence rates across age groups can be used in cross-sectional studies to estimate transmission rates in the absence of prospective sequential blood sampling, assuming a steady-state situation. In this study, we found the prevalence of each of the three markers of HBV infection to increase with age. Similar age trends have been observed in studies in Gambian and Cameroonian children [Whittle et al., 1983; Chiaramonte et al., 1991]. The increase in seroprevalence across age in this study supports the existence of frequent hoizontal transmission of HBV in children aged 6–18 years in the absence of sexual and parenteral exposures, as described by Davis et al. [1989]. We believe that almost all of the age-related increase in seroprevalence is likely to reflect the acquisition of new infection and is not due to a cohort effect, because factors that influence horizontal and vertical transmission have not significantly changed in the Ghanaian environment over the last 20 years. Hence, there is no reason to suspect an underlying difference in risk of exposure to HBV in children in different birth cohorts.

The age-related increase in HBV seroprevalence suggests an average incidence of new HBV infection of ~2.5% per year between the age of 6 years and 18 years in this district of Ghana. Like other studies [Sobeslavy et al., 1980; Davis et al., 1989], this study suggests that the risk of transmission of HBV might not uniform across age groups in children. The seroprevalence of those with any marker of HBV infection was relatively stable between 6 and 9 years, after which there was an increase in the rate until the 12–13-year age group (Table I). After this age, the prevalence of HBV infection remained constant with increasing age. HBsAg seroprevalence was estimated to be ~10% between 6- and 9-year-olds, after which it increased to ~21% by the age of 13 years, and thereafter remained ~15–20% (P value for trend 0.07). However, the relatively small numbers of children in each age cohort limits the conclusions that can be drawn from these data.

The age-related differences in HBsAg seroprevalence can be attributed to differences in the risk of acquiring HBV infection and the probability of remaining a HBsAg carrier once infected. Whittle et al. [1983] observed that HBsAg persisted over long periods in Gambian children, often well over 8 years after acquisition of the virus. Our methods did not permit determination of when the infection had been acquired. As a proxy for persistence of infection, we examined the rate of HBsAg carriage among those with HBV infection, which we defined as the proportion of anti-HBc positives who were also HBsAg positive ("carrier/infected rate"). This rate showed an increase from 9 years to a peak in 10–11-year-olds, after which it gradually fell. This pattern further

TABLE II. HCV Seroprevalence Rates in Various HBV Marker Groups in School and HBsAg Seroprevalence According to Anti-HCV Status

HBV marker status	Sample size	HCV seroprevalence (95% CI)	*P* value*
HBsAG positive	131	6.0 (2.2–9.7)	
HBsAg negative	672	5.3 (3.1–7.6)	0.63
Anti-HBc positive	468	5.6 (3.4–7.8)	
Anti-HBc negative	335	5.2 (2.0–8.3)	0.59
Anti-HBs positive	205	5.2 (2.3–8.1)	
Anti-HBs negative	588	5.6 (3.6–7.6)	0.42
Any HBV marker	516	5.4 (3.4–7.5)	
No HBV marker	287	5.4 (2.1–8.7)	0.99
HCV marker status		**HBsAg seroprevalence**	***P* value***
Anti-HCV positive	46	17.4 (5.2–29.6)	
Anti-HCV negative	755	15.4 (12.5–18.4)	0.77

**P* value is for test for significance difference between each pair of HBV and HCV marker status.

strengthens our hypothesis of a nonuniform rate of transmission in the age group studied.

The seroprevalence of "any HBV marker" and anti-HBc positivity was found to be significantly higher in males than in females. It is possible that these moderate gender differences may reflect more frequent high risk behaviors among males predisposing to horizontal transmission of HBV, such as fist fighting and other games involving close physical contact in this age group. However, it was surprising that the gender difference in HBsAg seroprevalence was smaller and not statistically significant.

The "carrier/infected rate's" in both males and females were estimated to be ~22% up to the age of 9 years, after which they rose to ~35–40% in the 10–11-year age group, then subsequently declined more in females than in males, until the age of 16–18 years when the rates in the two genders once more approximated each other (Fig. 1). This trend was also observed in Cameroonian children aged 4–14 years old, where a similar "carrier/infected rate" in males was estimated to be 23.3% compared to 16.6% in females [Chiaramonte et al., 1991]. The general decline in this rate after the 10–11-year age period in this study may be attributed to fewer new infections from horizontal transmission with increasing age as a result of fewer remaining susceptibles and reduced infectivity of HBsAg-positive individuals [Dazza et al., 1993]. However, the observation of a higher prevalence of HBV-DNA in males than in females in children as also described from Cameroon may indicate a higher replicative state in males than in females. Hence, infection may take a longer time to clear in males than in females, and this may possibly explain the difference in "carrier/infected rate's" observed in males and females.

Approximately 80% of patients recovering from acute HBV infection are normally thought to develop anti-HBs [Perrillo et al., 1991]. Kashiwagi et al. [1984] observed that when HBsAg seroprevalence increased from 17.2% in 0–9-year-old Japanese children to 42.6% in 10–19-year-olds, seroprevalence of anti-HBs also rose from 16.7% in 0–9-year-old HBsAg negative children to 96.3% in 10–19-year-olds in the same Japanese population, indicating a high rate of persistence of anti-HBs after clearance of HBsAg. However, in our study population, almost 55% (182 of 340) children who were both HBsAg-negative and anti-HBc positive did not have detectable anti-HBs. This trend was persistent in all the birth cohorts studied (Fig. 2). Also, whereas HBsAg seroprevalence increased from 10.1% to 20.9% and anti-HBc seroprevalence rose from 38.9% to 71.5% between ages 8–13, anti-HBs rose by only 8.3% (Table I). It is not clear why naturally acquired hepatitis B infection elicits such an infrequent and/or shortened anti-HBs response in this population. Potential antigen variation in strains of HBV prevalent in Ghana, other viral infections that might influence immune responses, the genetic characteristics of this population, and nutritional patterns in this population need to be evaluated to throw more light on this finding.

It was found that school children living in a rural district in Ghana also have an anti-HCV seroprevalence rate of 5.6%. This is similar to rates found in a recent study in Zaire [Tibbs et al., 1991], but much higher than the 1–2% obtained in a study of an urban South African population [Ellis et al., 1990] and much lower than the 16.5% seroprevalence found in a study of 400 Cameroonian school children [Ngatchu et al., 1992]. Hepatitis C virus infection has been described to be transmitted mainly through blood transfusions and parenteral drug abuse, with sexual and household contact representing relatively minor modes of transmission in western societies [Perrillo et al., 1991]. However, as many as 40% of infections occur in the absence of specific risk factors other than low socioeconomic factors. Although some Ghanaians receive multiple transfusions in early childhood, very few of such children are likely to survive to late childhood. Hence, very few children with repeated transfusions are likely to be present in the school age population we studied. The fact that such a high anti-HCV seroprevalence was found in a population that is not sexually active, not active intravenous drug users, and not likely to have been exposed to an unusually high number of blood transfusions suggests that casual

contact plays an important role in HCV transmission in this sub-Saharan African population [Hess et al., 1989; Hsu et al., 1991].

A general trend of increasing HCV seroprevalence was observed with age, consistent with casual contact being the predominant mode of transmission in these children. Most acute HCV infections are likely to be asymptomatic and result in chronic infection with seroconversion. However, acute nonpersisting infections do not elicit a life-long antibody response; consequently, seroprevalence includes very few of short-lived infections. As such, seroprevalence is not an indication of the fraction of the population ever infected. Since seroprevalence of the virus marker was relatively low in the study population, a slowly increasing trend in seroprevalence with age is consistent with long-term persistence of infection and antibody coupled with a relatively low rate of new infections. Although factors that influence HCV seroprevalence in this community are still being evaluated, no significant difference was found between males and females and between communities of various sizes. Similar observations were made in a study of Cameroonian school children [Ngatchu et al., 1992].

Limited evidence suggests that some chronic HBsAg carriers may undergo spontaneous clearance following superinfection with HCV [Liaw et al., 1991; Sheen et al., 1992], suggesting that HCV superinfection may suppress HBV. These observations suggest the possibility that HCV infection might modify HBV infection in high risk populations, although to date there are no epidemiologic data to support such a conclusion. HBV markers were found to be about 10 times more prevalent than HCV markers in this population. The prevalence of HBV markers did not vary according to HCV antibody status. Thus, there was no evidence that anti-HCV positive children are less likely to be HBsAg carriers because of HCV superinfection might suppress or terminate HBsAg carriage states in chronic HBV infections [Liaw et al., 1991; Sheen et al., 1992].

In conclusion, this study demonstrated a high prevalence of hepatitis B and C virus infection among school children in rural Ghana and reemphasizes the potential value of hepatitis B vaccine for this population. A high HBsAg carriage rate and a persistent low production of anti-HBs in those recovering from HBV infection were demonstrated and for the first time, we have detailed the patterns of HBsAg carriage and immune response to horizontally transmitted hepatitis B. These findings indicate a need for further evaluations of the consequence of a high rate of HBsAg carriage and the unusual immune response to HBV surface antigen (anti-HBs) in the context of horizontal transmission. The results of this survey reinforce the potential value of a vaccination program targeted at children in early life along with the other routine immunizations offered by the Expanded Programme on Immunization. HBV screening and possibly vaccination of adults thought to be at high risk should be secondary to a program targeting children, since resources in this environment are limited.

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